NOAA Pacific Islands Corals Science Workshop

CORAL DISEASE

June 18, 2012

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Hawaii Institute of Marine Biology

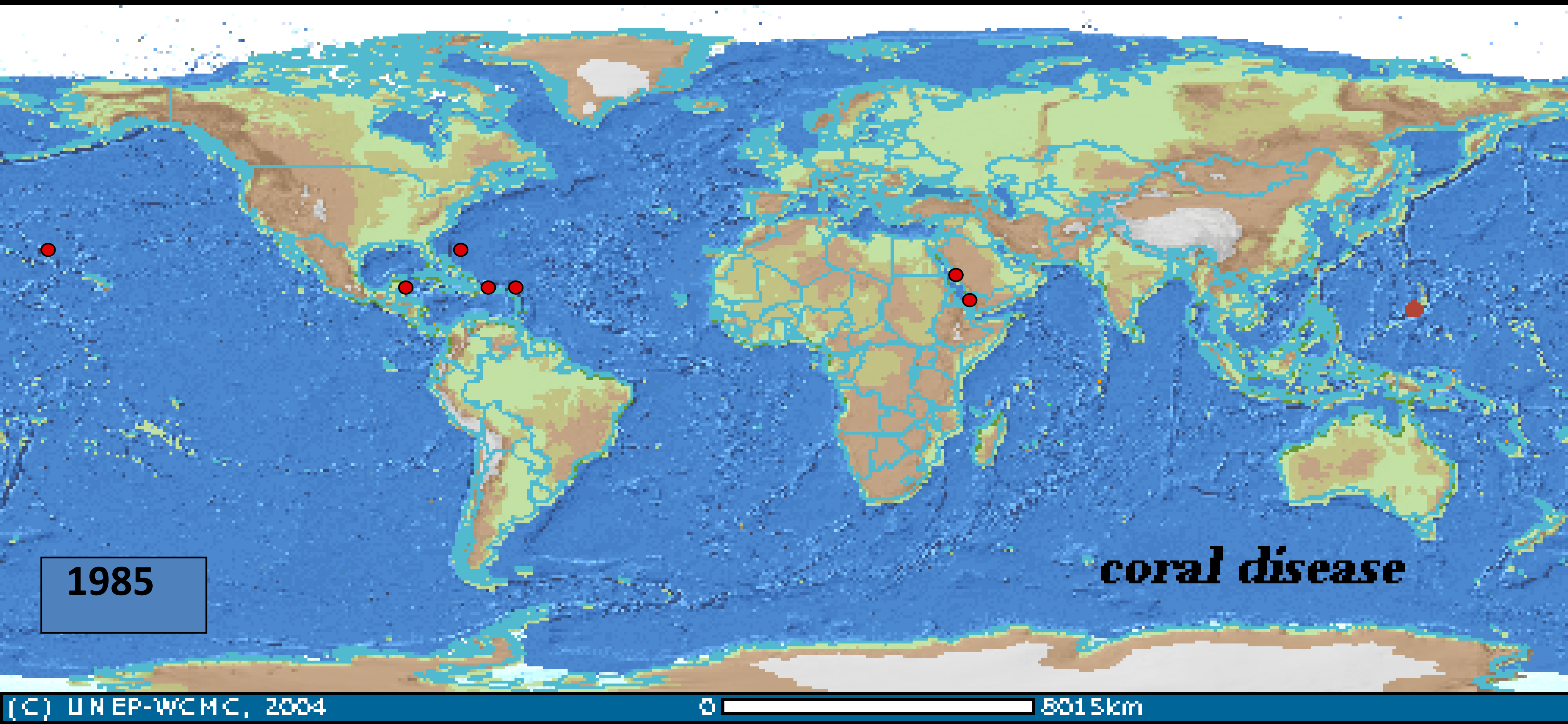
Coral Disease

Disease: Any impairment of vital body functions, systems, or organs.

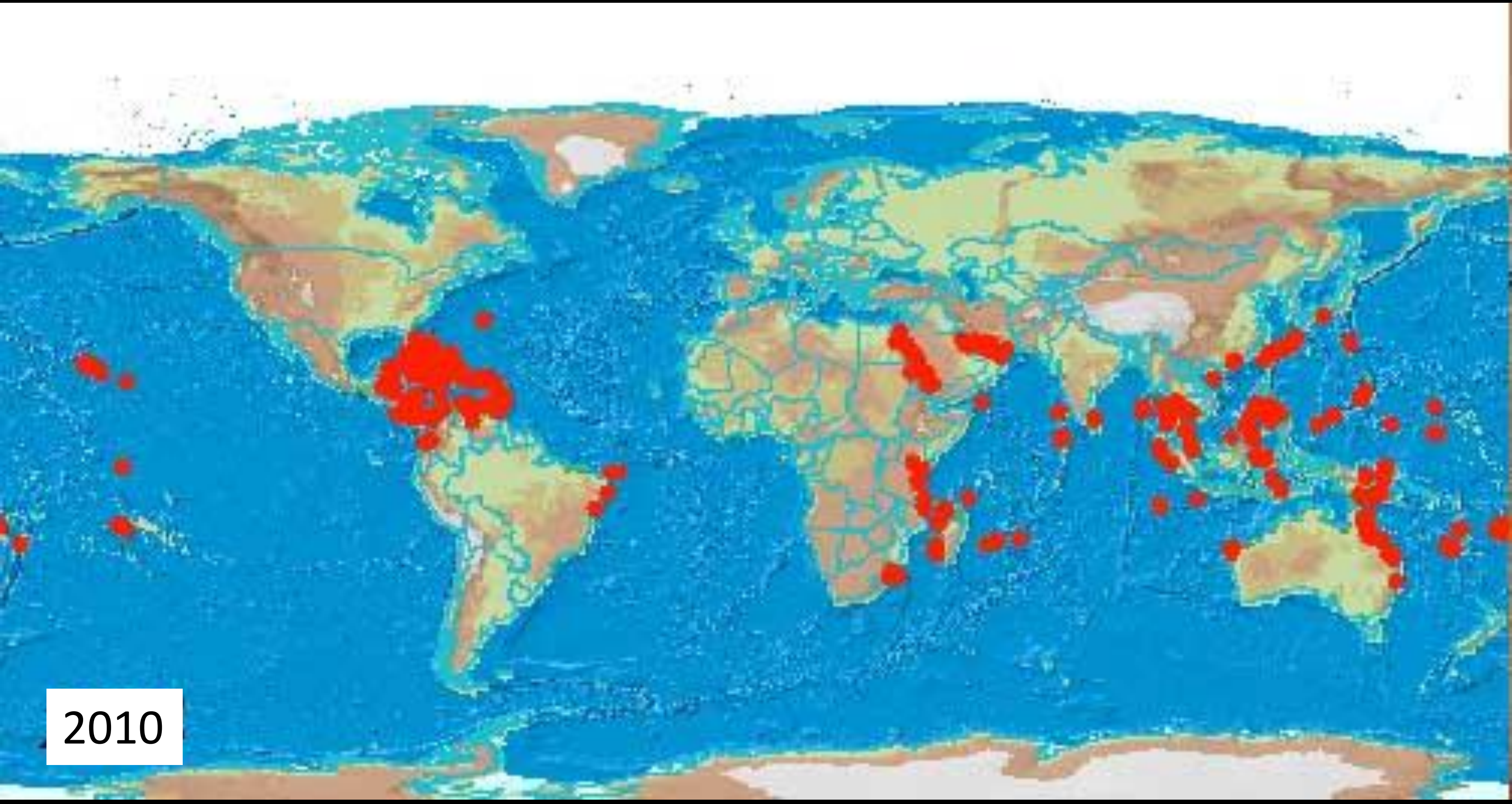
- **Biotic**
  - Causal agent a living organism
    - Pathogen,such as viruses or bacteria
    - Parasites

- **Abiotic**
  - Causal agent an environmental stressor
    - Changes in salinity, temperature, light, etc.
    - Exposure to toxic chemicals

Reports of coral disease across the world



Increases in distribution and abundance of coral disease



Aspergillosis

Florida Keys

1996-2000

 # stations w/ disease: 26 -> 131
 # coral species w/ disease: 11 -> 36
Overall coral cover: decreased by 37%

Porter et al. (2002)

Yellow band

White pox

Black band

Dark spots

Florida Keys

A. palmata

Losses of 87% or more

Patterson et al. 2002

Bahamas: Rainbow Reef

1991

Bahamas: Rainbow Reef

2004

Caribbean

Coral disease hotspot

Indo-Pacific

?

Aspergillosis

Yellow band

White pox

Black band

Dark spots

Lobophyllia white syndrome

Acropora white syndrome

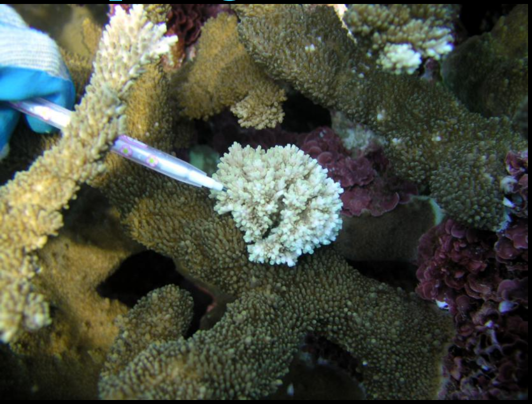
Australia
GBR

1998-2003

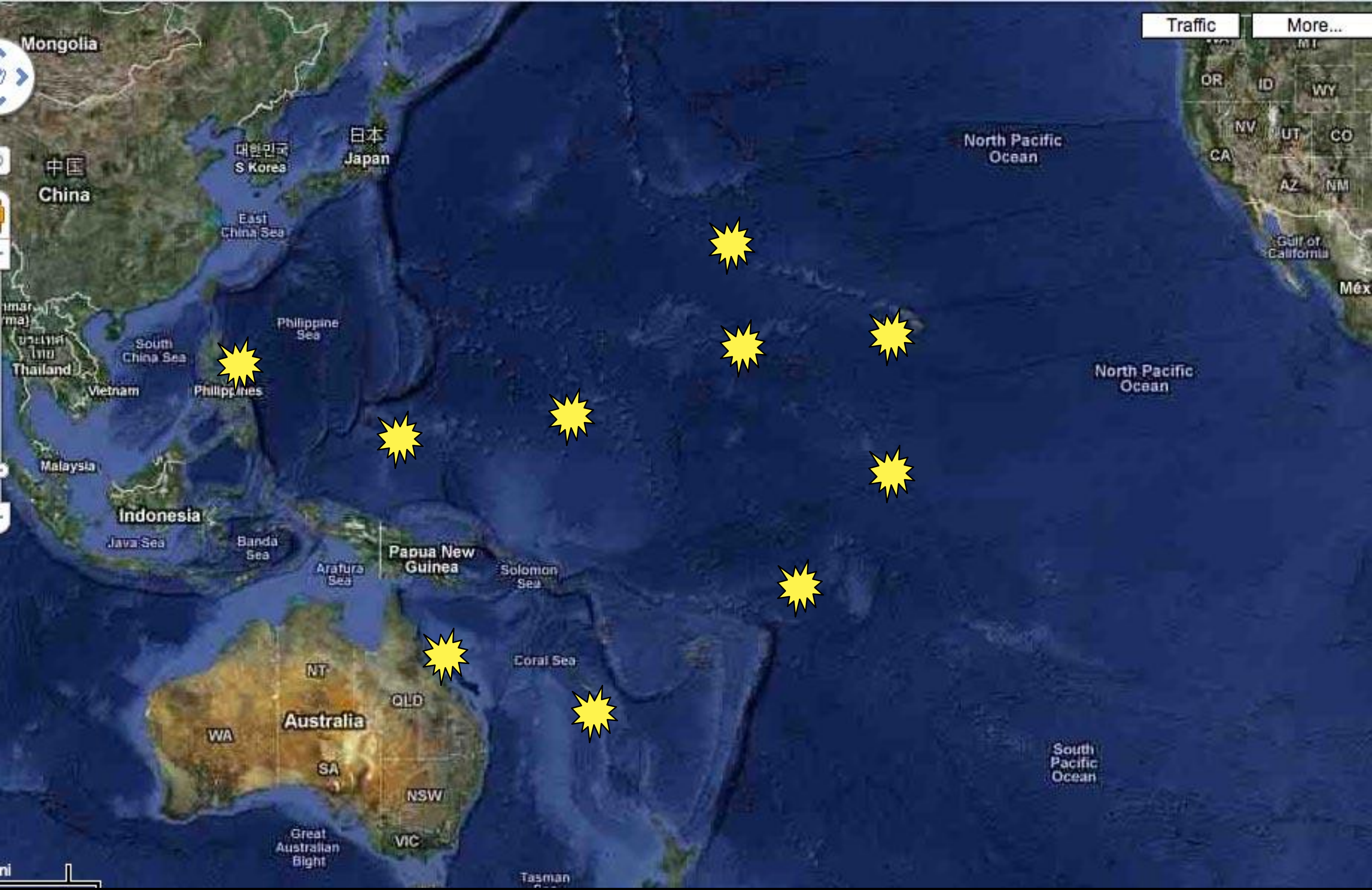
 # reefs w/ white syndrome: 4 -> 33
avg. # cases of white syndrome/reef: 1.7 -> 47.7

Willis et al. (2004)

Acropora growth anomalies



Black band



Disease outbreaks across the Indo-Pacific

CORAL DISEASE

Increasing worldwide

Caused extensive damage to reefs in W. Atlantic

Emerging as a problem in Indo-Pacific

Assessing threats from coral disease

- **Differential susceptibility among coral genera**
- **Virulence of different diseases**
- **Disease etiologies**
- **Predicted changes in disease threats with increasing anthropogenic stressors and global climate change**

Differential disease susceptibility among coral genera

Study	Region	coral genera
Willis et al. 2004	GBR	Pocilloporidae, Acroporidae, Poritidae
Haapkyla et al. 2010	GBR	*Acropora, Montipora*
Dalton & Smith 2006	eastern Australia	*Acropora, Turbinaria, Pocillopora*
Aeby et al. 2008	American Samoa	*Acropora, Pavona, Porites*
Myers & Raymundo 2009	Micronesia	*Porites, Acropora, Pocillopora*
Aeby et al. 2011	MHI & NWHI	*Porites, Acropora, Montipora*
Vargas-Angel 2009	US Pacific Remote Island Areas (Johnston Atoll, Wake, Baker, Howland, Jarvis, Palmyra, Kingman)	*Montipora, Porites, Acropora*
Raymundo et al. 2005	Philippines	*Porites*
Haapkyla et al. 2009	S.E. Sulawesi, Indonesia	*Acropora, Porites, Astreopora/Anacropora*
Hobbs & Frisch 2010	Indian Ocean Cocos Islands, Christmas Island	*Acropora*
Williams et al. 2011	Palmyra	*Astreopora, Acropora, Montipora*

Differential disease susceptibility among coral genera

Study	Region	coral genera
Willis et al. 2004	GBR	Pocilloporidae, Acroporidae, Poritidae
Haapkyla et al. 2010	GBR	*Acropora, Montipora*
Dalton & Smith 2006	eastern Australia	*Acropora, Turbinaria, Pocillopora*
Aeby et al. 2008	American Samoa	*Acropora, Pavona, Porites*
Myers & Raymundo 2009	Micronesia	*Porites, Acropora, Pocillopora*
Aeby et al. 2011	MHI & NWHI	*Porites, Acropora, Montipora*
Vargas-Angel 2009	US Pacific Remote Island Areas (Johnston Atoll, Wake, Baker, Howland, Jarvis, Palmyra, Kingman)	*Montipora, Porites, Acropora*
Raymundo et al. 2005	Philippines	*Porites*
Haapkyla et al. 2009	S.E. Sulawesi, Indonesia	*Acropora, Porites, Astreopora/Anacropora*
Hobbs & Frisch 2010	Indian Ocean Cocos Islands, Christmas Island	*Acropora*
Williams et al. 2011	Palmyra	*Astreopora, Acropora, Montipora*

Acropora, Montipora, Porites, Pocillopora, Astreopora, Turbinaria, Pavona

Coral Disease

- **Tissue loss**

- **Discoloration**

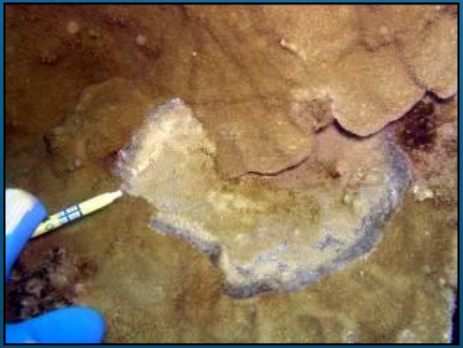
- **Growth anomaly**

Work & Aeby 2006

Coral Disease

Lesion Type | **Virulence**

- **Tissue loss**



Direct colony mortality

- **Discoloration**

Reduce coral growth
Decrease reproduction

- **Growth anomaly**

Rapid disease killing coral in Kaneohe Bay

Posted: Apr 02, 2010 10:38 AM
Updated: Apr 02, 2010 7:23 PM

By Jim Mendoza - bio | email

KANEOHE (HawaiiNewsNow) - In Kaneohe Bay the backbone of an ecosystem is under attack.

"What we're starting to see is whole clusters, ten, twenty, thirty colonies all dead in an area as the disease has passed from one other within the last four to five weeks," said Greta Aeby, a researcher with the Hawaii Institute of Marine Biology.

Acute Montipora White Syndrome -- a tissue killing slaughtered more than 100 colonies of red rice c Island.

"It usually comes in as a very bright whi colony. That white is where it's stripp said. "The problems here is that c you're wiping out a decade's wo

Most of the damage is in th quality's the poorest.

"Our reefs have be pollution for de the result of show for the sh homes there's no pl crumbles, she said. bigger guys,"

Aeby and other res zones and are analyzing coral samp

"One thing that we want hat may be causing this disease. So we're using histology, w microbiology, we're using molecular techniques to see might have been caused by a bacterial infection," she said.

LOCAL NEWS

Posted on: Monday, April 5, 2010

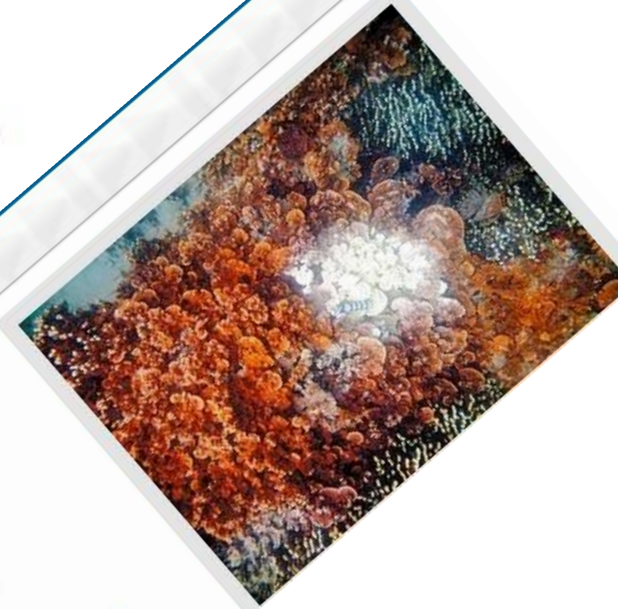
Disease hits Kāne'ohe Bay reefs

MWS has killed 100 colonies of red rice coral

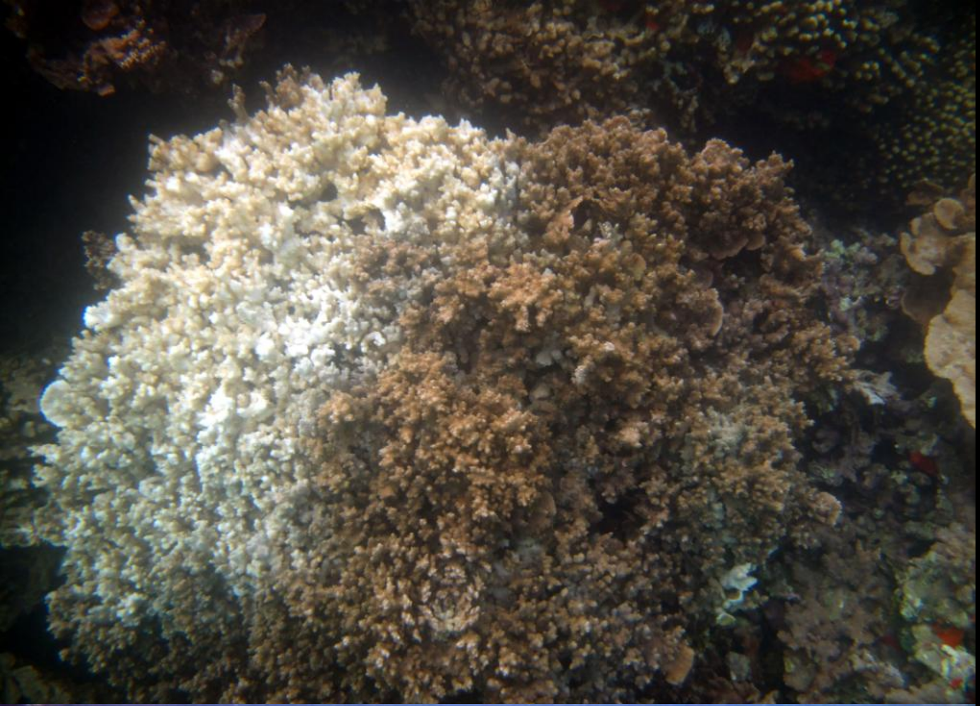
Comments (15) Recommend (1) Print this page E-mail this article Share

By John Windrow
Advertiser Staff Writer

Hawai'i scientists are battling a new threat to coral reefs in Kāne'ohe Bay that could imperil the biological balance in the bay's ecosystem.



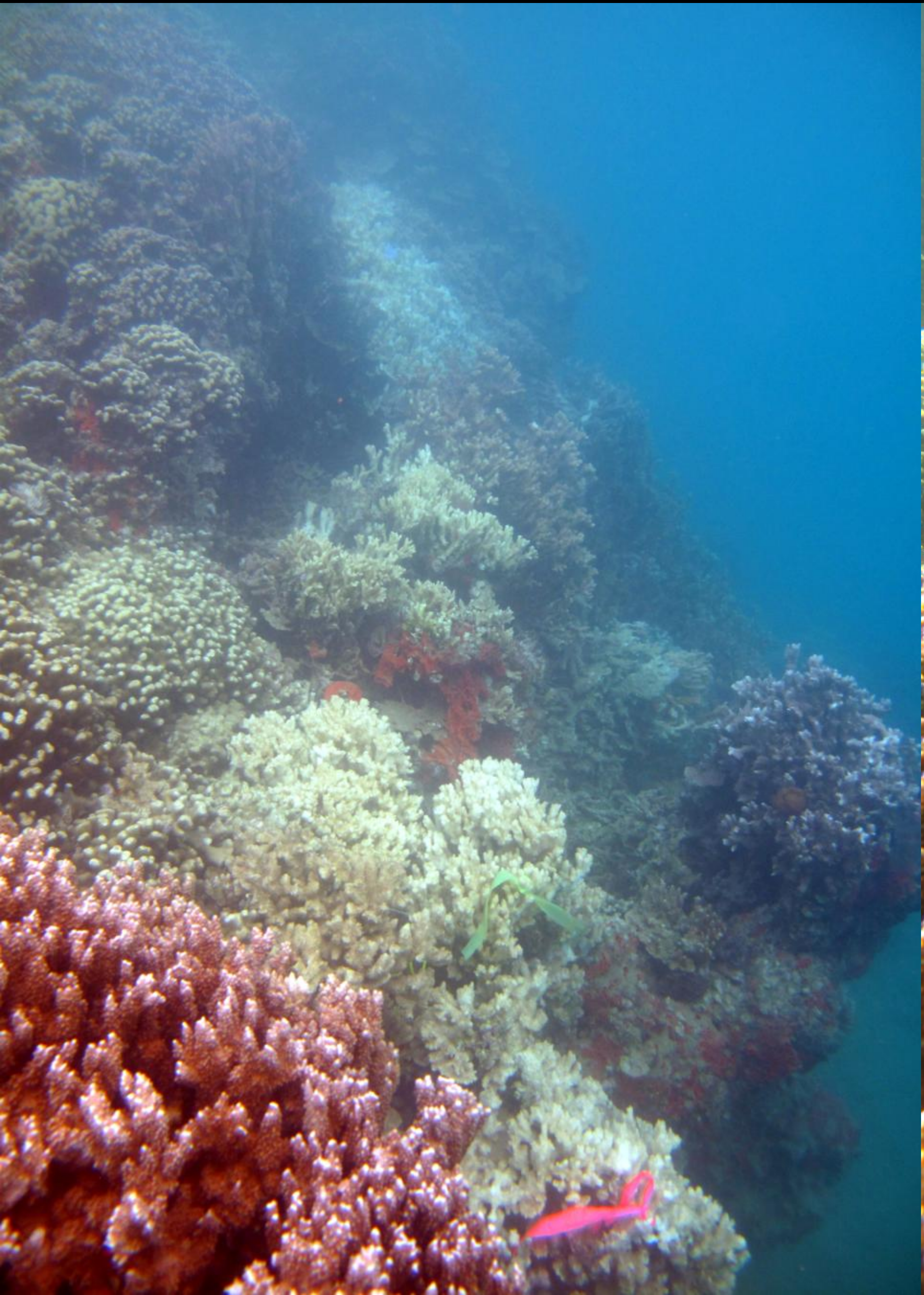
Acute *Montipora* White Syndrome



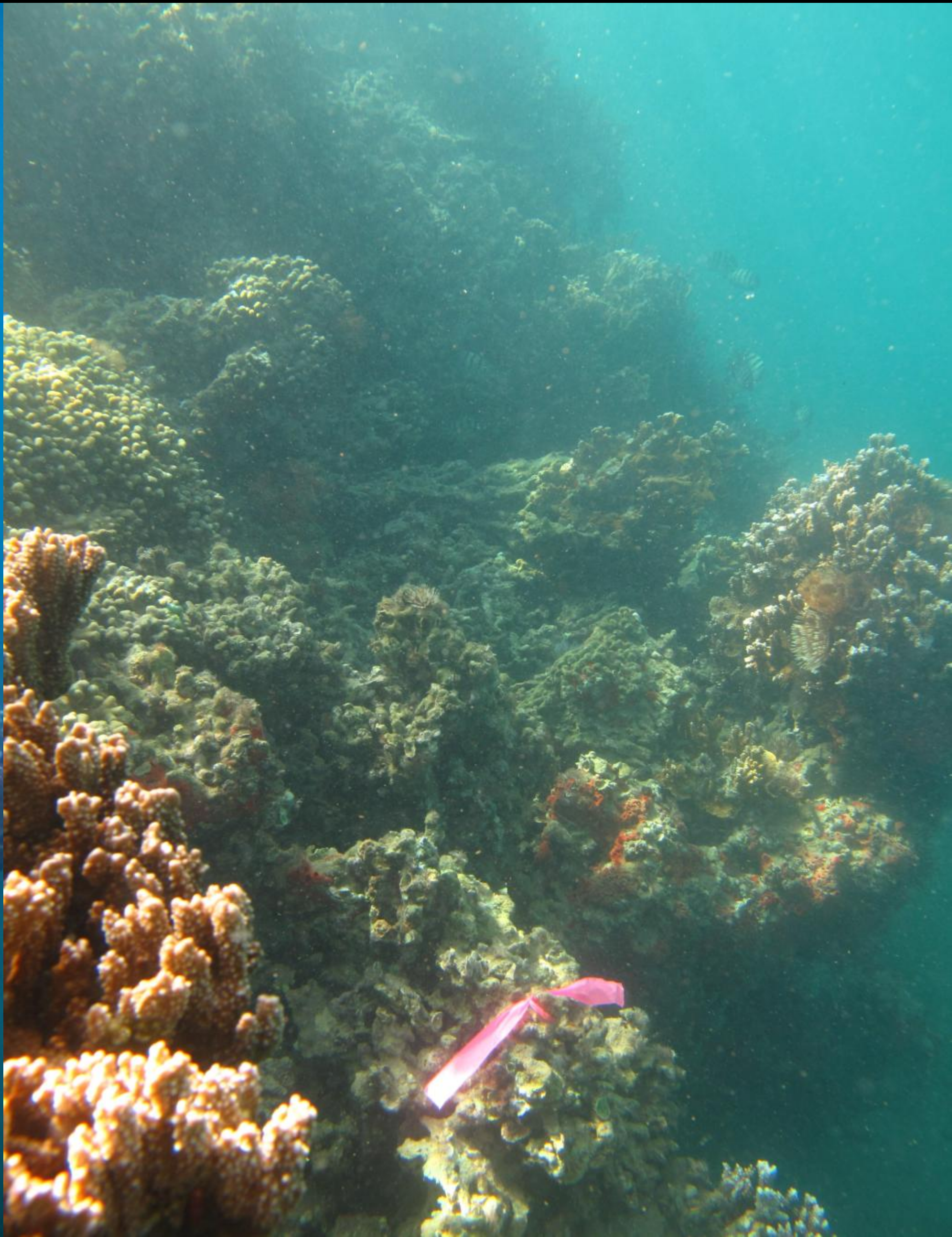
3-22-10

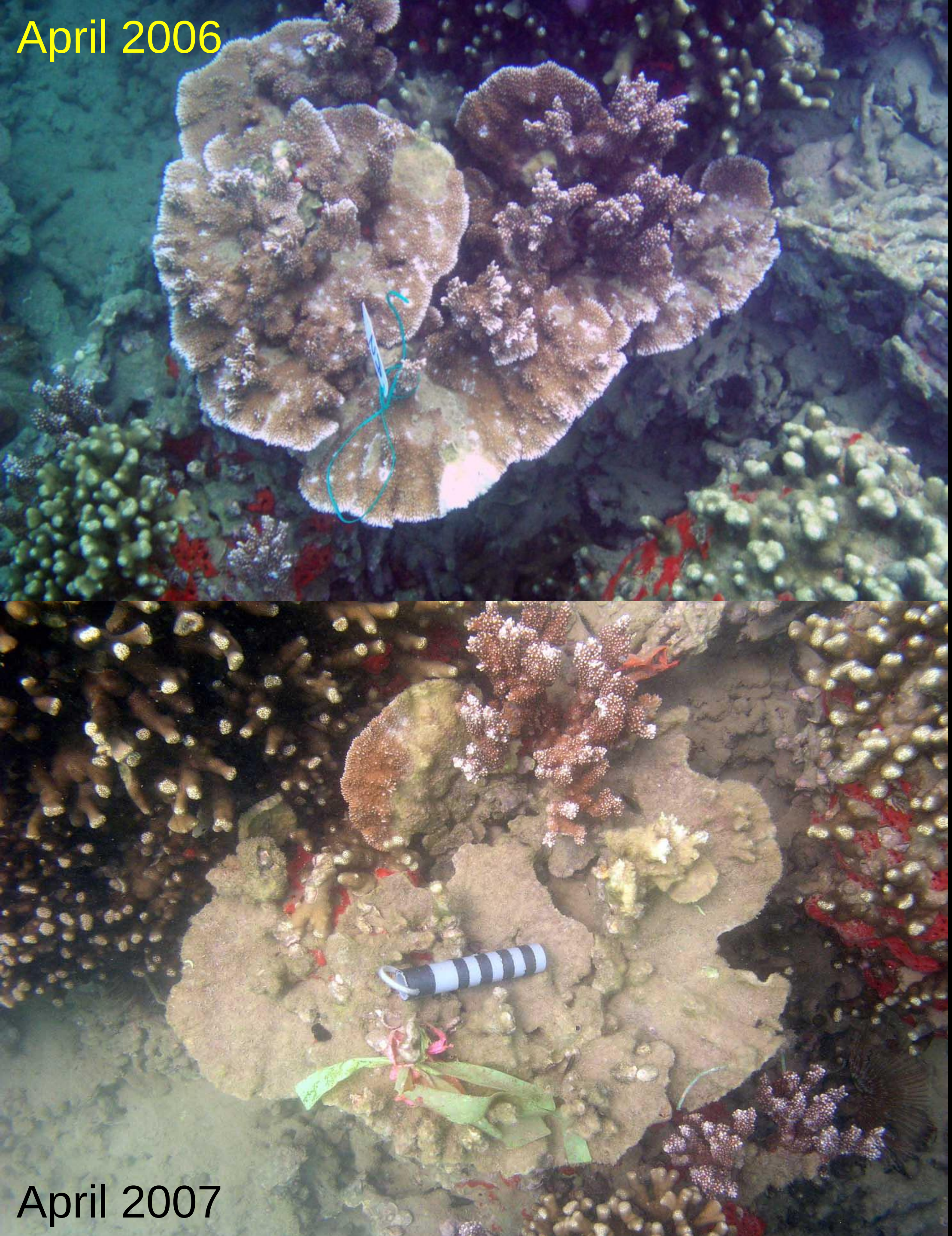
4-1-10

April 2010



April 2011





Chronic MWS

Rate of tissue lost:
~5% of colony/month

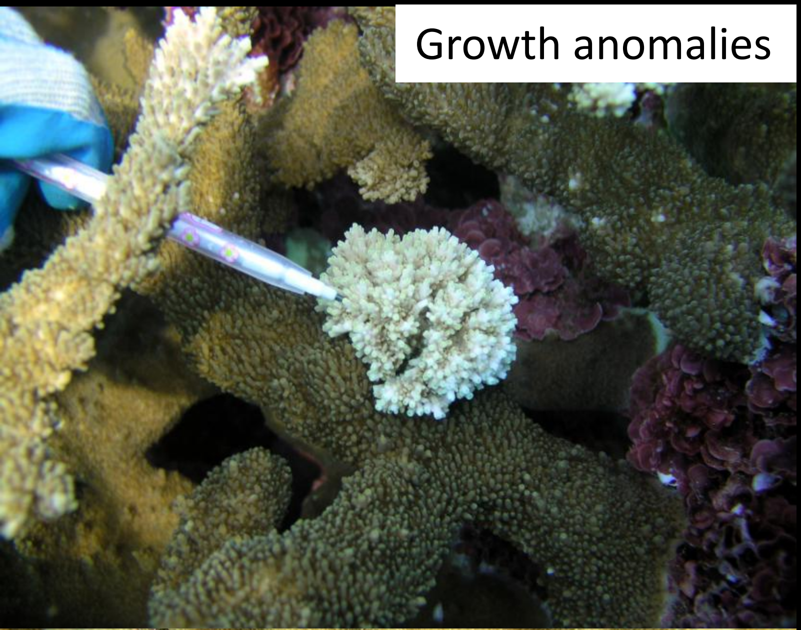
Tissue loss>90%:
~30% of colonies:

Tissue loss
Medium virulence

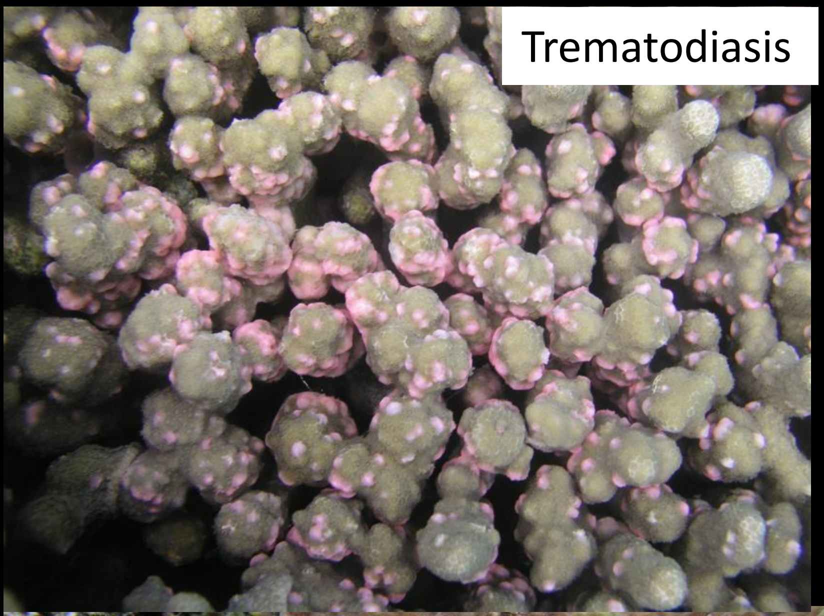
Acropora ciliate disease

Blackband disease

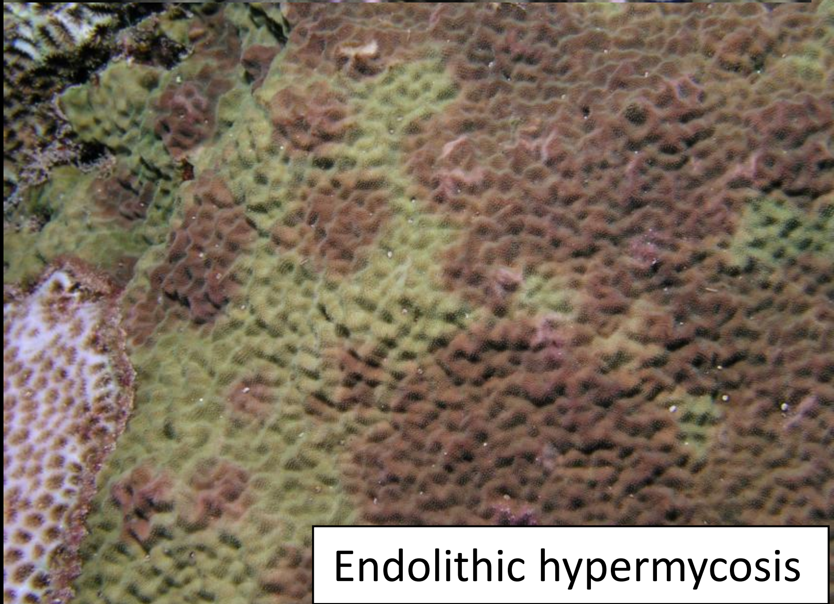
Low virulence



Growth anomalies



Trematodiasis



Endolithic hypermycosis

WS=white syndrome, a=acute, c=chronic, BBD=black band disease, SEB=skeletal eroding band, BrB=brown band, YBD=yellow band disease, mftl=multi-focal tissue loss, Bl=bleaching, GA=growth anomaly, DS=dark spot=endolithic hypermycosis, VCBl=*Vibrio coralliilyticus* induced bleaching, BND=brown necrotizing disease

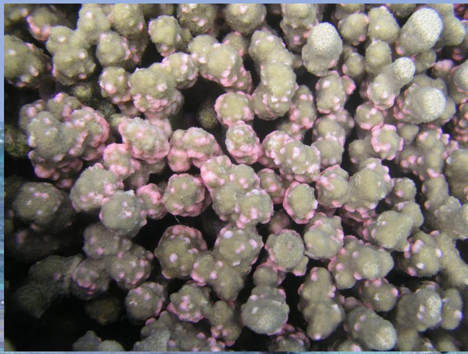
Coral genera	**high virulence: acute tissue loss**	**medium virulence: chronic tissue loss**	**low virulence: reduced growth or reproduction**
Acanthastrea			
Acropora	**aWS**	**cWS,BBD, ciliate, SEB, BrB, YBD**	**GA**
Alveopora			
Anacropora			
Astreopora	WS	SEB,BBD	GA
Barabattoia			
Caulastrea			
Cyphastreaa		SEB, YBD	
Euphyllia			
Galaxea	WS	SEB	
Isopora			
Leptoseris	WS	SEB	
Millepora		BBD, SEB	
Montipora	**aWS**	**cWS, mftl, BBD, SEB, ciliate, BrB**	**GA, DS**
Pachyseris			
Pavona	WS	SEB	GA, DS
Pectinia			
Physogyra			
Pocillopora	WS	SEB, BBD,BrB	GA, VCBl
Porites	**WS**	**mftl, cWS, BBD,PLS,YBD, BL w/ TS, BND, SEB**	**GA, trematodiasis**
Pssamocora			DS,
Seriatopora	WS	SEB,BBD	
Turbinaria	WS	mftl, BBD, YBD	GA

What can cause disease?

- virus
- bacteria
- fungus
- parasites
- protozoans
- chromosomal abnormalities
- abiotic

What can cause disease in coral?

- virus?
- bacteria
- fungus
- parasites
- protozoans
- algae
- abiotic?



PLoS one

Coral Pathogens Identified for White Syndrome (WS) Epizootics in the Indo-Pacific

Meir Sussman[1,2*], Bette L. Willis[1], Steven Victor[3], David G. Bourne[2]

1 ARC Centre of Excellence for Coral Reef Studies, School of Marine and Tropical Biology, James Cook University, Townsville, Australia, 2 Australian Institute of Marine Science (AIMS), PMB3, Townsville MC, Townsville, Australia, 3 Palau International Coral Reef Center (PICRC), Koror, Republic of Palau

Abstract

Background: White Syndrome (WS), a general term for scleractinian coral diseases with acute signs of advancing tissue lesions often resulting in total colony mortality, has been reported from numerous locations throughout the Indo-Pacific, constituting a growing threat to coral reef ecosystems.

Methodology/Principal Findings: Bacterial isolates were obtained from corals displaying disease signs at three WS outbreak sites: Nikko Bay in the Republic of Palau, Nelly Bay in the central Great Barrier Reef (GBR) and Majuro Atoll in the Republic of the Marshall Islands, and used in laboratory-based infection trials to satisfy Henle-Koch's postulates, Evan's rules and Hill's criteria for establishing causality. Infected colonies produced similar signs to those observed in the field following exposure to bacterial concentrations of 1×10^{6} cells ml^{-1}. Phylogenetic 16S rRNA gene analysis demonstrated that all six pathogens identified in this study were members of the γ-*Proteobacteria* family *Vibrionacae*, each with greater than 98% sequence identity with the previously characterized coral bleaching pathogen *Vibrio coralliilyticus*. Screening for proteolytic activity of more than 150 coral derived bacterial isolates by a biochemical assay and specific primers for a *Vibrio* family zinc-metalloprotease demonstrated a significant association between the presence of isolates capable of proteolytic activity and observed disease signs.

Conclusion/Significance: This is the first study to provide evidence for the involvement of a unique taxonomic group of bacterial pathogens in the aetiology of Indo-Pacific coral diseases affecting multiple coral species at multiple locations. Results from this study strongly suggest the need for further investigation of bacterial proteolytic enzymes as possible virulence factors involved in *Vibrio* associated acute coral infections.

Citation: Sussman M, Willis BL, Victor S, Bourne DG (2008) Coral Pathogens Identified for White Syndrome (WS) Epizootics in the Indo-Pacific. PLoS ONE 3(6): e2393. doi:10.1371/journal.pone.0002393

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Competing Interests: The authors have declared that no competing interests exist.

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Introduction

Reports on coral disease continue to rise [1] with currently 29 reported syndromes in the Caribbean [2] and 7 syndromes reported from the Indo-Pacific [3]. However, the causes for coral disease and the methods by which to investigate them are still heavily debated [4–6]. Most efforts are directed towards traditional surveillance [7], with comparatively less research directed towards developing strategies for active engagement in coral reef health management, disease prevention and cure [8–10]. Unfortunately, a lack of knowledge of coral disease causative agents propels this debate to a stand still. To date, only 5 bacterial species and one fungal agent have been determined as causative agents for coral infectious diseases [11–17], and currently no diagnostic tools or management efforts are able to validate these findings at a level required for active intervention. [18–19].

The study of disease in complex environmental settings is often difficult. Modern studies have cast a shadow on traditional culturing methods that are required to satisfy Henle-Koch's postulates [20], namely that a putative pathogen is first isolated on growth medium and then used in pure culture to duplicate disease signs in laboratory controlled infections. In many cases, more than 200 years after Henle-Koch's own revolution, these experiments often fail, requiring the introduction of modern rules and criteria in order to establish disease causation [21–22]. These are often based on statistical associations rather than on "cause and effect".

Most microorganisms cannot be easily cultured [23] and other disease components, namely host susceptibility and environmental factors may jointly contribute to successful infections in what is known as the "disease triad" [24]. To this end, modern diagnostic tools have been developed that can be applied to enhance our knowledge of coral disease without targeting either a single or a cultivable agent. These tools include cloning and denaturing gradient gel electrophoresis [25], fluorescent *in situ* hybridization [26], microarrays [27] and metagenomics [28–30], just to name a few, and are used to either detect new pathogens or validate their

White syndrome
Palau, Marshall Is, GBR

Vibrio coralliilyticus


Drivers of coral disease?

Coral bleaching and disease combine to cause extensive mortality on reefs in US Virgin Islands

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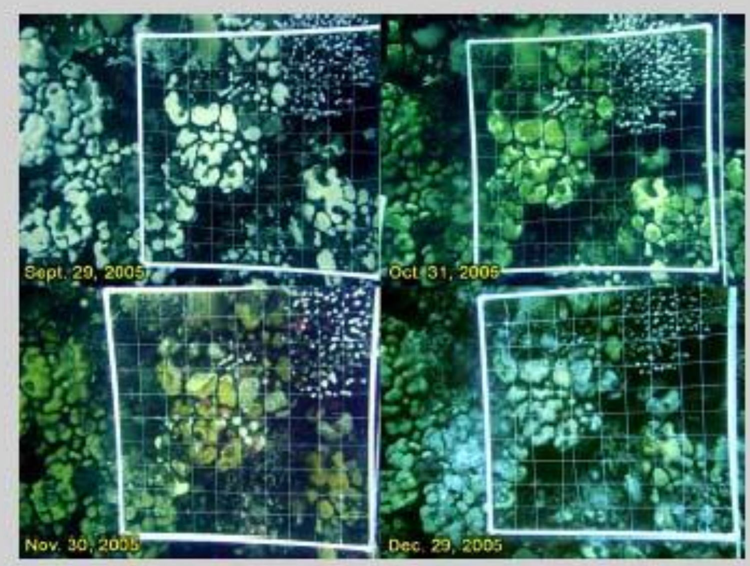

Fig. 1 Four-month time series of *Montastraea annularis* and *Porites porites*. Bleached condition on September 29, 2005. Partial color recovery by October 31, 2005. More color recovery but disease mortality on November 30, 2005. Mortality from disease spreads on December 29, 2005

Reefs in the US Virgin Islands experienced extensive bleaching in September 2005 with > 90% coral cover bleached (n = 20 video transects at each of five reef sites). Mean reef water temperatures from April 2005 to September 2005 were significantly higher than the previous 14 years. Corals began regaining color in October as water temperatures decreased, and minimal coral mortality was observed.

Monitoring from November 2005 to April 2006 revealed significant coral mortality following distinct White Plague disease signs, resulting in unprecedented 26–48% losses in coral cover (Fig. 1). Chronic mortality from this disease has occurred monthly at one monitoring site since 1997, but with prevalence rates not related to elevated temperatures or previous bleaching events (Miller et al. 2003).

While coral mortality from bleaching events has been well documented (Hoegh-Guldberg 1999) this study shows that only with frequent monitoring would these post-bleaching mortality patterns and presence of pathogenic disease be detected.

References
Hoegh-Guldberg O (1999) Climate change, coral bleaching and the future of the world's coral reefs. Mar Freshw Res 50:839–866
Miller J, Rogers C, Waara R (2003) Monitoring the coral disease, white plague on coral reefs in St. John US Virgin Islands. Rev Biol Trop 51(Suppl 4):47–55

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US Geological Survey, 1300 Cruz Bay Creek, St. John, VI 00380, USA

Reef sites

Coral Reefs (2006) 25: 418
DOI 10.1007/s00338-006-0125-6

Sept 2005

- >90% coral bleached
- White plague outbreak followed in November 2005
- 26-48% loss of coral cover

Thermal Stress and Coral Cover as Drivers of Coral Disease Outbreaks

John F. Bruno[1*], Elizabeth R. Selig[2], Kenneth S. Casey[3], Cathie A. Page[4], Bette L. Willis[4], C. Drew Harvell[5], Hugh Sweatman[6], Amy M. Melendy[7]

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Very little is known about how environmental changes such as increasing temperature affect disease dynamics in the ocean, especially at large spatial scales. We asked whether the frequency of warm temperature anomalies is positively related to the frequency of coral disease across 1,500 km of Australia's Great Barrier Reef. We used a new high-resolution satellite dataset of ocean temperature and 6 y of coral disease and coral cover data from annual surveys of 48 reefs to answer this question. We found a highly significant relationship between the frequencies of warm temperature anomalies and of white syndrome, an emergent disease, or potentially, a group of diseases, of Pacific reef-building corals. The effect of temperature was highly dependent on coral cover because white syndrome outbreaks followed warm years, but only on high (>50%) cover reefs, suggesting an important role of host density as a threshold for outbreaks. Our results indicate that the frequency of temperature anomalies, which is predicted to increase in most tropical oceans, can increase the susceptibility of corals to disease, leading to outbreaks where corals are abundant.

Citation: Bruno JF, Selig ER, Casey KS, Page CA, Willis BL, et al. (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biol 5(6): e124. doi:10.1371/journal.pbio.0050124

Introduction

Climatic and oceanographic conditions can modify a wide variety of ecological processes. For example, ocean temperature can control species ranges, the strength of species interactions, the dispersal and survival of marine larvae, and the rates of metabolism and speciation [1–6]. Additionally, anomalously high temperature and other environmental stresses can influence the severity and dynamics of infectious diseases by increasing host susceptibility and pathogen virulence [7,8]. For example, the severity of human epidemics including cholera [9–11] and tick-borne encephalitis [12] are both related to temperature and, possibly, to recent climate change [13]. Temperature and climate change have also been implicated in plant and animal disease outbreaks in both terrestrial and aquatic habitats [7,14–17], and could influence coral disease severity [18–20], potentially accelerating the global loss of coral reefs.

Corals are the foundation species of tropical coral reef ecosystems. They directly facilitate thousands of associated species by generating the physically complex reef structure [21,22]. Reductions in coral abundance can cause rapid loss of reef biodiversity [23]. The hypothesized link between anomalously high temperatures and coral disease outbreaks is supported by small-scale field studies indicating that prevalence and the rate of within-colony spread of several coral diseases are higher during the summer [24–30]. Such seasonal changes in disease severity could be driven in part by higher summertime temperature, but could also be caused by a variety of other abiotic factors that vary seasonally within sites. Additionally, such investigations do not directly address the role of temperature anomalies in driving the conspicuous variability of coral disease severity among years and locations [30–32] that has long intrigued coral reef ecologists. Missing are large-scale, longitudinal investigations that combine long-term monitoring of multiple populations with accurate, fine-grained measurements of local temperature anomalies. Longitudinal studies (i.e., the repeated sampling of individuals or populations) help control for potential confounding factors and inherent temporal variability [33]. Such powerful epidemiological approaches are rarely applied to marine epidemics (but see [34,35]), which has limited our understanding of potential links between temperature and disease outbreaks in the ocean, especially at large spatial scales.

Here we describe a regional-scale test of the hypothesis that ocean temperature can influence disease frequency. We analyzed the relationship between the frequency of white syndrome in scleractinian corals and of warm temperature anomalies across the Great Barrier Reef (GBR). Forty-eight reefs were monitored for 6 y (1998–2004), and reef-specific

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Abbreviations: GBR, Great Barrier Reef; OISST, Optimum Interpolation Sea Surface Temperature; SST, sea surface temperature; WSSTA, weekly sea surface temperature anomaly

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Host abundance
Thermal stress

OPEN ACCESS Freely available online

PLoS one

Growth Anomalies on the Coral Genera *Acropora* and *Porites* Are Strongly Associated with Host Density and Human Population Size across the Indo-Pacific

Greta S. Aeby[1]*, Gareth J. Williams[2,3]*, Erik C. Franklin[1], Jessica Haapkyla[4], C. Drew Harvell[5,6], Stephen Neale[4], Cathie A. Page[4], Laurie Raymundo[7], Bernardo Vargas-Ángel[8], Bette L. Willis[4], Thierry M. Work[9], Simon K. Davy[2]

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Abstract

Growth anomalies (GAs) are common, tumor-like diseases that can cause significant morbidity and decreased fecundity in the major Indo-Pacific reef-building coral genera, *Acropora* and *Porites*. GAs are unusually tractable for testing hypotheses about drivers of coral disease because of their pan-Pacific distributions, relatively high occurrence, and unambiguous ease of identification. We modeled multiple disease-environment associations that may underlie the prevalence of *Acropora* growth anomalies (AGA) (n = 304 surveys) and *Porites* growth anomalies (PGA) (n = 602 surveys) from across the Indo-Pacific. Nine predictor variables were modeled, including coral host abundance, human population size, and sea surface temperature and ultra-violet radiation anomalies. Prevalence of both AGAs and PGAs were strongly host density-dependent. PGAs additionally showed strong positive associations with human population size. Although this association has been widely posited, this is one of the first broad-scale studies unambiguously linking a coral disease with human population size. These results emphasize that individual coral diseases can show relatively distinct patterns of association with environmental predictors, even in similar diseases (growth anomalies) found on different host genera (*Acropora* vs. *Porites*). As human densities and environmental degradation increase globally, the prevalence of coral diseases like PGAs could increase accordingly, halted only perhaps by declines in host density below thresholds required for disease establishment.

Citation: Aeby GS, Williams GJ, Franklin EC, Haapkyla J, Harvell CD, et al. (2011) Growth Anomalies on the Coral Genera *Acropora* and *Porites* Are Strongly Associated with Host Density and Human Population Size across the Indo-Pacific. PLoS ONE 6(2): e16887. doi:10.1371/journal.pone.0016887

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Competing Interests: The authors have declared that no competing interests exist.

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Introduction

Coral reefs represent some of the most biologically diverse ecosystems on the planet, but these important habitats are declining worldwide due to human overexploitation, land-based pollution, global climate change, and disease outbreaks [1–6]. While the situation is most severe in the Caribbean, coral reefs are also in decline across the Indo-Pacific, where an annual loss in coral cover of approximately 1% has occurred over the last 20 years, increasing to 2% between 1997 and 2003 [7]. Coral diseases contribute to this decline by causing a loss of live coral cover [8–10] that, under extreme circumstances, can lead to complete community phase-shifts (e.g. from coral-dominated to alga-dominated) [11]. The causes of most coral diseases are unknown. However, understanding how coral disease prevalence relates to changes in reef environmental quality may provide clues to disease etiology. Coral disease increases are associated with local anthropogenic stressors such as poor water quality [12–17], as well as global stressors such as sea-surface temperature anomalies [18] and resultant coral bleaching events [19–22]. Effects of environmental co-factors may vary between disease types [23] but few efforts have been made to model individual coral diseases with multiple, possibly interacting, environmental cofactors, but see [17,18,22].

Host abundance

Humans

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REPORT

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Nutrient enrichment enhances black band disease progression in corals

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Abstract Infectious diseases are recognized as significant contributors to the dramatic loss of corals observed worldwide. However, the causes of increased coral disease prevalence and severity are not well understood. One potential factor is elevated nutrient concentration related to localized anthropogenic activities such as inadequate waste water treatment or terrestrial runoff. In this study the effect of nutrient enrichment on the progression of black band disease (BBD) was investigated using both in situ and laboratory experiments. Experimental increases in localized nutrient availability using commercial time release fertilizer in situ resulted in doubling of BBD progression and coral tissue loss in the common reef framework coral *Siderastrea siderea*. Laboratory experiments in which artificially infected *S. siderea* colonies were exposed to increased nitrate concentrations (up to 3 μM) demonstrated similar increases in BBD progression. These findings provide evidence that the impacts of this disease on coral populations are exacerbated by nutrient enrichment and that management to curtail excess nutrient loading may be important for reducing coral cover loss due to BBD.

Keywords Coral disease · Black band disease · Nutrient enrichment · Coral reefs

Introduction

Globally, coral reefs are undergoing detrimental loss and degradation (Hughes et al. 2003). This trend has been particularly severe on reefs of the wider Caribbean, where an estimated 80% of coral cover has been lost over the past three decades (Gardner et al. 2003). Concurrent with losses in coral cover there have been dramatic increases in the number, frequency, geographic distribution, and host range of coral diseases (Richardson 1998; Harvell et al. 1999; Porter et al. 2001; Richardson and Aronson 2002; Sutherland et al. 2004; Weil 2004). Infectious diseases of corals are now recognized as significant contributors to the degradation observed in coral communities, particularly on Caribbean reefs (Rosenberg and Loya 2004). To date, 18 coral diseases have been described (Sutherland et al. 2004), but only six of these have been characterized in terms of pathogen identification and disease etiology.

Although the reasons for the increases in coral disease incidence and prevalence are largely unknown (Richardson et al. 1998), numerous factors including both natural and local anthropogenic impacts have been suggested as potential contributors to this phenomenon (Jackson et al. 2001; Rosenberg and Ben-Haim 2002). Of particular concern is anthropogenic nutrient enrichment. Potential human-related sources of elevated nutrients to the reef environment include inadequate sewage treatment (Lapointe et al. 1990; Paul et al. 1995; Szmant and Forrester 1996; Lipp et al. 2002), and increased terrestrial runoff related to development (Hallock et al. 1993; Costa et al. 2000). The most common natural, offshore contributions to nutrient enrichment originate from coastal upwelling, occasionally associated with internal tidal bores (Leichter et al. 1996, 2003) or volcanic events (Genin et al. 1995). Both increased nutrient availability and decreased herbivory can lead to phase shifts from coral-dominated to macroalgal-dominated communities (Littler and Littler 1984; Done 1992; Hughes 1994; Steneck and Dethier 1994). Only a limited number of studies have assessed the potential interactions between increased nutrients and reef degradation other than phase shifts. One potential effect may be an enhancement of pathogen-associated coral diseases.

To date, four published studies have included a quantitative assessment of the relationship between

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Nutrient stress

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PLoS one

Seasonal Rainfall and Runoff Promote Coral Disease on an Inshore Reef

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Abstract

Background: Declining water quality coupled with the effects of climate change are rapidly increasing coral diseases on reefs worldwide, although links between coral diseases and environmental parameters remain poorly understood. This is the first study to document a correlation between coral disease and water quality on an inshore reef.

Methodology/Principal Findings: The temporal dynamics of the coral disease atramentous necrosis (AN) was investigated over two years within inshore populations of *Montipora aequituberculata* in the central Great Barrier Reef, in relation to rainfall, salinity, temperature, water column chlorophyll *a*, suspended solids, sedimentation, dissolved organic carbon, and particulate nitrogen, phosphorus and organic carbon. Overall, mean AN prevalence was 10-fold greater during summer wet seasons than winter dry seasons. A 2.5-fold greater mean disease abundance was detected during the summer of 2009 (44 ± SE 6.7 diseased colonies per 25 m^2), when rainfall was 1.6-fold greater than in the summer of 2008. Two water quality parameters explained 67% of the variance in monthly disease prevalence in a Partial Least Squares regression analysis; disease abundance was negatively correlated with salinity ($R2 = -0.6$) but positively correlated with water column particulate organic carbon concentration ($R2 = 0.32$). Seasonal temperature patterns were also positively correlated with disease abundance, but explained only a small portion of the variance.

Conclusions/Significance: The results suggest that rainfall and associated runoff may facilitate seasonal disease outbreaks, potentially by reducing host fitness or by increasing pathogen virulence due to higher availability of nutrients and organic matter. In the future, rainfall and seawater temperatures are likely to increase due to climate change which may lead to decreased health of inshore reefs.

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Introduction

Disease has emerged as a significant threat to wildlife populations in recent decades [e.g.1, 2]. A recent review highlights the substantial role that environmental nutrient enrichment has played in contributing to patterns of emerging human and wildlife diseases and the urgent need for studies to understand linkages, particularly in light of ongoing intensification of global nutrient cycles [3]. The current understanding of marine diseases is poor in comparison to knowledge of human, agricultural and terrestrial wildlife diseases [4]. It appears that epidemiological theories developed for terrestrial diseases may not translate well to marine ecosystems [4,5]. For example, diseases appear to spread more rapidly in comparatively open oceanic ecosystems [6] and marine pathogens are more diverse taxonomically and in their life histories [5]. Thus, marine case studies that advance understanding of potential links between nutrient enrichment and marine diseases are critical if management tools for the long-term conservation of marine wildlife are to be effective.

Coral reefs are increasingly threatened by changes in water quality from terrestrial runoff [7], climate change [8,9] and over-exploitation [10,11]. Coral bleaching and disease have emerged as dominant drivers of coral population declines on coral reefs, particularly as oceans have warmed in the past few decades [12]. Current research supports a connection between a warming climate and increasing incidence of disease in corals [12,13,14]. For example, warm temperatures and high coral cover have been linked to increased abundance of white syndromes on the Great Barrier Reef (GBR) [14] and progression rates of black band disease were higher in the austral summer [15,16]. However, links to most other anthropogenic disturbances are less clear [17].

Although the mechanisms are unknown, outbreaks of disease on some coral reefs have been correlated with increases in nutrient runoff [18,19]. In the Philippines, a higher prevalence of growth anomalies and *Porites* ulcerative white spot disease was found near a sewage outfall [20], and white pox has also been linked to sewage inputs in the Caribbean [21]. Field experiments in the Caribbean have demonstrated that moderate increases in dissolved inorganic

Land-based sources of pollution

Drivers of coral disease

Host abundance

Global climate change
- increased sea surface temperature
- increased temperature anomalies
- increased frequency of bleaching events

Human stressors
- Land-based pollution
- Sedimentation
- Overfishing
- Human usage

Assessing threats from coral disease

Differential susceptibility among coral genera

•*Acropora, Montipora, Porites, Pocillopora, Turbinarea, Astreopora, Pavona*

Virulence of different diseases

•Acute tissue loss – chronic tissue loss – reduced growth or reproduction

Disease etiologies

•Bacteria, fungus, parasites, protozoa

Predicted changes in disease threats with increasing anthropogenic stressors and global climate change

•Increased frequency of disease outbreaks

•Increased virulence